UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/512,124	08/26/2005	Genhong Cheng	02307K-154600US	8432

20350 7590 09/06/2007

TOWNSEND AND TOWNSEND AND CREW, LLP
TWO EMBARCADERO CENTER
EIGHTH FLOOR
SAN FRANCISCO, CA 94111-3834

EXAMINER	
DANG, IAN D	
ART UNIT	**PAPER NUMBER**
1647	

MAIL DATE	DELIVERY MODE
09/06/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/512,124	CHENG ET AL.	
	Examiner	Art Unit	
	Ian Dang	1647	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *10 July 2007*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-24* is/are pending in the application.

4a) Of the above claim(s) *18,19 and 22-24* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-17,20 and 21* is/are rejected.

7)☒ Claim(s) *1* is/are objected to.

8)☒ Claim(s) *1-24* are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *10/20/2004* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Election/Restrictions

Applicant's election with traverse of Group I, claims 1-17 and 20-21 in the communication filed on 07/10/2007 is acknowledged. Applicant has further elected the species TLR3 and poly I:C in the communication filed on 07/10/2007. The traversal for the election of Group I is on the ground(s) that the 3 groups set forth by the examiner stem from a common concept and theory and are thus related. As such prosecution of the claims of Groups I-IV would not place a substantially greater burden on the Examiner.

This is not found persuasive because the inventions listed as Groups I-IV do not relate to a single general inventive concept under PCT Rule 13.1 because, under PCT Rule 13.2, they lack the same or corresponding special technical features for the following reasons:

According to PCT Rule 13.2, unity of invention exists only when the shared same or corresponding technical feature is a contribution over the prior art. The inventions listed as Groups I-IV do not relate to a single general inventive concept because they lack the same or corresponding technical feature.

Claim 1 is directed to a method for activating interferon regulatory factor 3 (IRF3) in a cell comprising contacting the cell with a molecule that stimulates a Toll-like receptor (TLR). Kawai et al., (Published November 15, 2001, The Journal of Immunology, Volume 167, Issue 10, page 5887-5894) teach a method for activating interferon regulatory factor 3 (IRF3) in a cell comprising contacting the cell with the TRL ligand lipopolysaccharide (LPS) that stimulates the Toll-like receptors (TLR) 4, thereby activating the IRF3 in the cell, wherein the cell expresses the TLR. The prior art meets the limitations disclosed in claim 1. Thus Group I lacks novelty or inventive step and does not make a contribution over the prior art. Since the first claimed

invention has no special technical feature, it cannot share a special technical feature with the other claimed invention.

Under PCR Rule 13.1, the application shall relate to one invention only or to a group of inventions so linked as to form a single general inventive concept.

The requirement is still deemed proper and is therefore made FINAL.

The election of species for the species of TRL and the species of TRL ligand as set forth in the in Restriction Election of 15 May 2007 is withdrawn, since there is no search burden on the Examiner.

Claims 18-19 and 22-24 are withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to a nonelected invention, there being no allowable generic or linking claim.

Claims 1-17, 20, and 21 are pending and under examination.

Specification

The disclosure is objected to because of the following informalities:

The disclosure is objected to because it contains an embedded hyperlink and/or other form of browser-executable code (see page 7, line 20; page 31, line 29; page 37, line 28). Applicant is required to delete the embedded hyperlink and/or other form of browser-executable code. See MPEP § 608.01.

Appropriate correction is required.

Claim Objections

Claim 1 is objected to because of the following informalities: Claim 1, line 2 recites "a Toll-like receptors". The term "receptors" should be made singular. Appropriate correction is required.

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 1-17 and 20-21 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

i. The phrase "stimulates induction of IRF3" in claims 5, 6, 7, 8, 10-17 is a relative phrase which renders the claims indefinite. The phrase "stimulates induction of IRF3" is not defined by the claims, the specification does not provide a standard for ascertaining the requisite degree, and one of ordinary skill in the art would not be reasonably apprised of the scope of the invention. It is not clear what biological effects encompassed by induction of IRF3. For instance, the induction of IRF3 could be the phosphorylation of IRF3, the translocation of IRF3, or the binding of IRF3 to gene promoters.

ii. Claim 11 recites the limitation "primary response protein" in line 1. There is insufficient antecedent basis for this limitation in the claim. It is noted that claim 11 depends from claim 6, which recites "primary response genes" and not "primary response protein".

iii. Claims 14 and 15 are rejected because claim 14 recites the limitation "expression of IFNβ" in line 1. There is insufficient antecedent basis for this limitation in the claim. It is noted

that claim 14 depends from claim 13, which recites "inducing activity of IFNβ" and not "expression of IFNβ".

iv. Claim 13 is indefinite because the claims have a step that does not clearly relate back to the preamble. For example, the preamble of claim 13 recites "a method for inhibiting a microbial infection." However, there is no step in the body of the claim indicating that the inhibition of a microbial infection has taken place.

v. The phrase "inducing activity of IFNβ" in claim 13 is a relative phrase which renders the claim indefinite. The phrase "inducing activity of IFNβ" is not defined by the claim, the specification does not provide a standard for ascertaining the requisite degree, and one of ordinary skill in the art would not be reasonably apprised of the scope of the invention. It is not clear what are biological effects encompassed by inducing activity of IFN.

vi. The term "stimulate" in claims 1-3, 4, 7-10, 20, and 21 is a relative term which renders the claim indefinite. The term "stimulate" is not defined by the claims, the specification does not provide a standard for ascertaining the requisite degree, and one of ordinary skill in the art would not be reasonably apprised of the scope of the invention. It is not clear what are biological effects encompassed by "stimulate". For instance, "stimulate" may mean dimerization, phosphorylation, or translocation of a TLR.

vii. The phrase "TLR3/TLR4 and IRF3 pathways" in claims 20 and 21 is a relative term which renders the claim indefinite. The phrase " TLR3/TLR4 and IRF3 pathways" is not defined by the claim, the specification does not provide a standard for ascertaining the requisite degree, and one of ordinary skill in the art would not be reasonably apprised of the scope of the invention. It is not clear what molecules are encompassed by the phrase.

viii. Claim 4 recites the limitation "binds the TLR" in line 1. There is insufficient antecedent basis for this limitation in the claim. It is noted that claim 4 depends from claim 1, which recites "stimulates a TLR" and not "binds the TLR".

ix. Claim 17 recites the limitation "stimulates activity of IRF3" in line 1. There is insufficient antecedent basis for this limitation in the claim. It is noted that claim 17 depends from claim 13, which recites "stimulates induction of IRF3" and not "stimulates activity of IRF3".

x. The terms "IRF3" "TLR", "IFNβ", "TRL3/TRL4" in claims 5-17, 20-21 are relative terms which render the claims indefinite. The terms "IRF3" "TLR", "IFNβ", "TRL3/TRL4" are not defined by the claim, the specification does not provide a standard for ascertaining the requisite degree, and one of ordinary skill in the art would not be reasonably apprised of the scope of the invention. For instance, claims 5-17, 20-21 use acronyms without first defining what they represent in the independent claims. While the claims can reference acronyms, the material presented by the acronym must be clearly set forth at the first use of the acronym.

xi. The phrase "activating the IRF3" in claim 1 is a relative phrase which renders the claim indefinite. The phrase "activating the IRF3" is not defined by the claim, the specification does not provide a standard for ascertaining the requisite degree, and one of ordinary skill in the art would not be reasonably apprised of the scope of the invention. It is not clear what are the biological activities encompassed by the activation of IRF3.

Claim Rejections - 35 USC § 112 (Written Description)

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 20-21 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Claim 20 is drawn to a method for inhibiting viral replication in a cell by stimulating the TLR3/TLR4 and IRF3 pathways in the cell comprising contacting the cell with a molecule that stimulates the TLR3/TLR4 and IRF3 pathways, thereby inhibiting the viral replication in the cell.

The specification does not provide any identifying characteristics for discerning the TLR3/TLR4 and IRF3 pathways recited in claim 20.

Thus, the claims are genus claims. The specification and claims do not indicate what distinguishing attributes are shared by the members of the genus. Specifically, the specification does not clearly define the TLR3/TLR4 pathway, the IFR3 pathway and all methods of using such. Thus, the scope of the claims includes numerous structural and functional variants, and the genus' are highly variant because a significant number of structural and functional differences between genus members is permitted. The specification and claims do not provide any guidance as to what changes should be made. Structural and functional features that could distinguish the TLR3/TLR4 pathway and the IFR3 pathway are missing from the disclosure. No common attributes identify the members of the genus. The general knowledge and level of skill in the art do not supplement the omitted description because specific, not general, guidance is what is needed. Since the disclosure fails to describe the common attributes or characteristics that identify members of the genus, and because the genus is highly variant, the TLR3/TLR4 pathway and the IFR3 pathway are insufficient to describe the genus.

The written description requirement for a claimed genus' may be satisfied through sufficient description of a representative number of species by actual reduction to practice, reduction to drawings, or by disclosure of relevant identifying characteristics, i.e. structure or other physical and/or chemical properties, by functional characteristics coupled with a known or disclosed correlation between structure and function structure, or by a combination of such identifying characteristics, sufficient to show the applicant was in possession of the claimed genus. In the instant case, the specification fails to provide sufficient descriptive information, such as definitive structural or functional features of the genus of the TLR3/TLR4 pathway and the IFR3 pathway and all methods of using such.

There is no description of the special features, which are critical to the structure and function of the genus claimed. Furthermore, the prior art does not provide compensatory structural or correlative teachings sufficient to enable one of skill to isolate and identify the TLR3/TLR4 pathway and the IFR3 pathway encompassed by the claims. Thus, no identifying characteristics or properties of the instant TLR3/TLR4 pathway and IFR3 pathway are insufficient to describe the genus are provided such that one of skill would be able to predictably identify the encompassed variant biological and chemical entities recited in the methods of the instant claims. One of skill in the art would reasonably conclude that the disclosure fails to provide a representative number of species to describe the genus. Thus, applicant was not in possession of the claimed genus.

Claim Rejections - 35 USC § 112 (Enablement)

Claims 5-17, 20-21 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not

described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

In In re Wands, 8USPQ2d, 1400 (CAFC 1988) page 1404, the factors to be considered in determining whether a disclosure would require undue experimentation include: (1) Nature of the invention, (2) the state of the prior art, (3) the predictability or lack thereof in the art, (4) the amount of direction or guidance present, (5) the presence or absence of working examples, (6) the breath of the claims, (7) the quantity of experimentation needed, (8) relative skill of those in the art.

Nature of the invention and breath of the claims

The invention is drawn to a method for activating interferon regulatory factor 3 (IRF3) in a cell comprising contacting the cell with a molecule that stimulates a Toll-like receptor (TLR), thereby activating the IRF3 in the cell, wherein the cell expresses the TLR. The invention is broad because the recitations of claims 5-7,11-16, and 20 encompass a large number of Toll-like receptors, molecules, IRF3 cellular responses, and cells.

For instance, the specification teaches that Toll-like receptors (TRL) refers to Toll-like receptors which play a critical role in innate immunity by recognizing structurally conserved pathogen associated molecular patterns (PAMPs) (page 13, lines 9-11). In addition, the specification teaches that the molecules or agents that suppress stimulation of TLR include but are not limited to a soluble TLR (page 16 lines 19-20). In addition, the specification teaches that molecules that block an endotoxin shock, such as an anti-LPS antibody may used to suppress stimulation of TLR. Additionally, molecules that block interaction of TLR with PAMP, such as an anti-PAMP antibody, may be used to suppress stimulation of TLR. Finally, the specification teaches that activation refers to the cellular changes in response to an environmental stimulus,

resulting in activity of a multitude of biochemical signaling pathways and significant changes in gene expression (page 14, lines 15-17).

Finally, the specification teaches that the only cells used to investigate the activation of IRF3 include murine bone marrow-derived macrophages (BMMs), the RAW 264.7 murine macrophage cells (page 32), and B-cells (page 35). There application does not study in the activation of IRF3 in any other cell types.

Thus the one of skill in the art is not enabled to make or use the claimed invention because the specification does not provide sufficient guidance for the use of the genus of Toll-like receptors; molecules, and IRF3 cellular responses for the claimed invention.

Unpredictability and state of the art

The state of the art for the expression of genes induced by the activation of IRF3 and TLR3 is well known, but the roles and mechanisms of IRF3 and TLR3 in innate immune response have not been established at this point. Several recent studies including one by the inventors of the instant application have indicated that the role of IRF3 in innate immune response is unpredictable.

For instance, Doyle et al. (2002, Immunity, Volume 17, page 251-63) teach that the role of IRF3 or IFNβ in bacterial infection is not well understood (page 260, left column 2nd paragraph). In addition Doyle et al. (2002) teach that more work must be done to determine the true functional outcomes of TLR3/TRL4 activation (page 260, left column, 2nd paragraph). In addition, in post-filing date literature, Doyle et al. (2003) teach that it remains unsolved how IRF3 becomes activated following TLR3 or TLR4 receptor stimulation (page 3570, right column, last paragraph). Finally, Alexopoulou et al. (2001, Nature, Volume 413, pages 732-738) teach that the importance of TLR3 in the antiviral response, however, remains to be established (page

738, left column, top paragraph). Thus one skilled in the art would not be able to reliably predict the roles of IRF3 and TLR3 in innate immune response.

The amount of direction or guidance present

Applicants' disclosure is limited to the identification and validation of a subset of genes following IRF3 activation, the identification of antiviral genes expression following TLR3 activation (page 49), and the protein expression profile of bone marrow-derived macrophages following the exposure of these cells to the bacteria *listeria monocytogenes* (LM). However, the specification does not provide guidance or direction regarding (1) a method for inhibiting a microbial infection, (2) a method for inhibiting a microbial infection by inducing activity of IFNβ in a cell, and (3) a method for inhibiting viral replication in a cell by stimulating the TRL3/TLR4 and IRF3 pathways in a cell. In addition, the specification does not provide guidance regarding the identifying characteristics of molecules that can stimulate any TLR, inhibits microbial infection, inhibits viral replication in a cell, and stimulates the TRL3/TRL4 and IRF3 pathways.

While the specification recites that IRF3 and TLR3 mediate the expression of numerous genes, the specification has not provided any guidance regarding the link between the expression of these genes and their functions in an innate immune function. Thus Applicant has not provided any guidance for the physiologic roles of these expressed IRF3 mediated proteins during an innate immune response, such as a viral or microbial infection.

In addition, the specification has not provided guidance regarding the relationship between the activity of IFNβ during a microbial infection and the stimulation of IRF3. Finally, the specification does not provide any direction with respect to specific proteins activated in the TLR3/TLR4 and IFR3 pathways and their roles in inhibiting viral replication in a cell. In addition, although the specification discloses the inhibition of MHV68 replication in macrophages

following TLR3 and TLR4 activation (page 41), the art is silent regarding the roles of IRF3 and TLR3 in a method for inhibiting viral replication in a cell.

Working Examples

Although Applicants have provided an example identification of a subset of genes induced by stimulation of TLR3 and demonstrated that IRF3 are key transcription factors response for this gene expression (for Example 1, page 43), identification of antiviral genes expression following TLR3 activation (Example 2, page 49), the protein expression following the exposure of bone marrow-derived macrophages in response to the bacteria *listeria monocytogenes* (LM) (Example 5, page 60), and the specification also does not provide any methods or working examples for (1) a method for inhibiting a microbial infection, or (2) a method for inhibiting a microbial infection by inducing activity of IFNβ in a cell. For instance, the specification provides one working examples using an *in vitro* or *in vivo* model for the inhibition of a microbial infection with a molecule that has been validated to activate IRF3.

Moreover, while the specification provides an example for the inhibition of MHV68 replication in macrophages following TLR3 and TLR4 activation (page 41), there is no other working examples for a method for inhibiting other viral replication in other cells by stimulating the TLR3/TLR4 and IRF3 pathways in the cell.

The quantity of experimentation needed

Without sufficient disclosure in the specification, it would require undue experimentation for one of skill in the art to be able to activate IRF3 in a cell with any molecules because the specification has not provided the identifying structural characteristics of the molecule necessary for activating IRF3.

In addition, it would require undue experimentation to practice the invention commensurate in scope with the claims because, the claims are broadly drawn (1) a method for activating interferon regulatory factor 3 (IRF3) in a cell comprising contacting the cell with a molecule that stimulates a Toll-like receptors (TLR), thereby activating the IRF3 in the cell, wherein the cell expresses the TLR, (2) a method for inhibiting a microbial infection by inducing activity of IFNβ in cell, and (3) a method for inhibiting viral replication in a cell by stimulating the TLR3/TLR4 and IRF3 pathways in the cell.

Finally, a large quantity of experimentation is required to inhibite a microbial infection, such as a bacterial infection, because the roles of IRF3 and TLR3 as disclosed by the inventors during an innate immune response have not been established. For instance, Doyle et al. (2002, Immunity, Volume 17, page 251-63) teach that the role of IRF3 or IFNβ in bacterial infection is not well understood (page 260, left column 2nd paragraph).

Therefore, the specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make/use the claimed methods of the instant application.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 1, 3, 4, 13, and 17 are rejected under 35 U.S.C. 102(a) as being anticipated by Kawai et al., (Published November 15, 2001, The Journal of Immunology, Volume 167, Issue 10, page 5887-5894)

Kawai et al. teach a method for activating interferon regulatory factor 3 (IRF3) in a cell comprising contacting the cell with the TRL ligand lipopolysaccharide (LPS) that stimulates the Toll-like receptor (TLR) 4, thereby activating the IRF3 in the cell, wherein the cell expresses the TLR (page 5887, abstract) meeting the limitations of claims 1, 3, 4, 13, and 17.

Claim 20 and 21 are rejected under 35 U.S.C. 102(a) as being anticipated by Kawai et al., (Published November 15, 2001, The Journal of Immunology, Volume 167, Issue 10, page 5887-5894).

The reference by Kawai et al. teach that the molecule LPS binds to TLR4 leading to the stimulation of the TLR3/TLR4 pathway and activates IRF3 leading to the simulation of the IRF3 pathway (page 5887, abstract) meeting the limitations of claims 20 and 21. Although the reference by Kawai et al. does not teach the inhibition of viral replication, the stimulation of the TLR3/TLR4 and IRF3 pathways would inherently result in the inhibition of the of viral replication. It is noted that a compound and all of its properties are inseparable; they are one and the same thing (see *In re Papesch*, CCPA 137 USPQ 43; *In re Swinehart and Sfiligoj*, 169 USPQ 226 (CCPA 1971)).

Claims 1-4, and 13 are rejected under 35 U.S.C. 102(b) as being anticipated Navarro et al. (1999, The Journal of Biological Chemistry, Volume 274, Number 50, pages 35535-35538). Navarro et al. teach a method comprising activating IRF3 by contacting the cell with the TRL ligand lipopolysaccharide (LPS), resulting in the activation or induction of IRF3 in the cell (page

35535, abstract) meeting the limitations of claims 1-4, and 13. It is noted that Navarro et al., teach that Toll-like receptors confer sensitivity toward LPS (page 35535, column 2, 2nd full paragraph) and that the prior art recognized that LPS activates Toll-like receptors (see Yang et al., Nature 395:284-288, 1998 and Qureshi et al., J.Exp. Med 189:615-625, 1999; as cited in Navarro et al.). In addition, Navarro et al. teach that IRF3 has been found to be an important cellular response to viral infection (page 35535, right column, first paragraph) and IRF3 is known to participate in the transcriptional induction of interferon beta genes (page 35535, abstract). Thus the reference by Navarro et al. teach the limitations encompassed by claim 13.

Conclusion

No claim is allowed.

Prior Art

The prior art is made of record and not relied upon is considered pertinent to Applicants' disclosure.

Alexopoulou et al. (2001, Nature, Volume 413, pages 732-737) teach that poly(I:C) binds to the TLR receptor TLR3.

Information

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Ian Dang whose telephone number is (571) 272-5014. The examiner can normally be reached on Monday-Friday from 9am to 5pm. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Manjunath Rao can be reached on (571) 272-0939. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300. I

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Ian Dang
Patent Examiner
Art Unit 1647
August 30, 2007

Bridget E. Bunner

BRIDGET E. BUNNER
PRIMARY EXAMINER